SAFETY FACTORS IN SPINAL ANESTHESIA *

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TO J. Leonard Corning of New York is usually given the credit of being the first to apply the principles of spinal anesthesia. In 1888 he injected cocain into the extradural space for the relief of symptoms in four cases of spinal disease. In 1899 August Bier of Bonn used the same principle and first conducted regional anesthesia by using the same drug in a similar fashion. Like many other new discoveries in medical arts, it was soon tried by the profession, but one by one physicians became discouraged and dropped the method because of serious and fatal complications to this procedure; the toxicity of cocain and lack of refinement of technique were responsible. In 1903 Fourneau discovered the less toxic stovain, and this was soon used instead of cocain. Numerous further refinements have been accomplished after many years of costly experimentation until we have now reduced the disadvantages and risks of this form of anesthesia to a minimal degree.

Numerous reports of large series of spinal anesthesias are being made from surgical clinics here and abroad. Each series shows a higher degree of safety to the patient than other forms of major anesthesia. Spinal anesthesia is apparently on the increase. Yet each writer invariably notes a few casualties, and remarks about some of the untoward or dangerous sequelae to this form of anesthesia. Babcock [1] states that from the depression of the respiration and the great fall of blood pressure which it causes, it has the largest potential danger. He even states that "in the hypotension of shock, hemorrhage, toxemia, or asthenia, while the skillful associated use of adrenalized intravenous injections may reduce its dangers, it remains unsafe for general use."

We are not here decrying the use of spinal anesthesia because of its dangers; on the contrary, in reasonably skillful hands the risk from this form of anesthesia should be very slight indeed. Yet while there are accepted real or potential dangers to this method, it is the purpose of this paper to point out these dangers and to offer suggestions for combating them.

PHYSIOLOGIC ACTION

To interpret the unusual happenings during an operation under spinal anesthesia necessitates an understanding of the physiologic background.

Since the spinal cord is surrounded by a fluid medium it is obvious that when drugs of anesthetizing property are injected into this fluid, there will result a local area of such influence. Hence the sensory, sympathetic and motor nerves which come in contact with the anesthetizing solution promptly absorb it and as promptly lose their power of conductivity. Anesthesia, sympathetic and motor paralysis follow in the segments corresponding to the segment represented at the point of injection. The spinal cord itself is little affected.

Within a few moments after injection the patient begins to note a loss of sensation in the lower extremities, followed in succession by motor weakness, and at times by an absolute motor paralysis if the anesthesia is carried that far. The sensory nerves are first affected and their conductivity much more interfered with than those of motor origin; when weak or insufficient drugs are used anesthesia can be established without interference with motor function. Tactile sensation is also preserved even after anesthesia and motor paralysis.

After the extremities have become paralyzed the abdominal muscles are similarly affected; the abdomen loses its normal contour and becomes more flat. The abdominal muscles cease to partake of the function of breathing.

To visualize what is happening in the meantime necessitates attention to the abdominal and thoracic nervous system. Recall that the sympathetic nervous system has communication with the spinal cord by sympathetic bundles which travel with the roots and later branch away and are then designated as rami communicantes. These sympathetic fibers proceed to the abdominal organs via several ganglia and are largely distributed to the blood vessels of the abdomen and thorax; they are vasoconstrictor fibers and give to the circulation that quality known as tonicity. The sympathetic system has a further function of being the motor accelerator of the thoracic organs, while in the abdomen the same system has a motor depressor function. Quite apart from the sympathetic system is the vagus nerve with its extensive distribution to the thorax and abdomen. Its impulses for activity are directly opposed to those of the sympathetic, since it has a depressor action in the thorax while it supplies the motor accelerator impulses to the organs below the diaphragm.

When the anesthetic solution interrupts the conductivity of spinal roots, it is obvious that the sympathetic fibers carried there will also be deadened. That means that the sympathetic control to the abdomen and thorax will be lost, while the vagus control comes more prominently in relief. Clinically we see several things as a result of this. First there is a sudden and tremendous drop in *blood pressure;* the splanchnic vessels having lost their tonicity dilate and become a reservoir for a large part of the blood in circulation. Peripheral blood gravitates to the dependent portions, and if we observe closely we note that there is a pallor of the uppermost part of the body while lividity can be noted dependently. In extreme cases the radials become pulseless to the touch and may not even record with the sphygmomanometer. If the patient has the head elevated or is in a sitting position, he begins to note giddiness and may follow on to syncope or coma simply because the cerebral blood has flowed into the splanchnics; the volume of the blood cannot be suddenly increased, hence peripheral circulation must suffer, and this is particularly true in the brain, where blood vessels do not have the support of firm surrounding structures as they have in other parts of the body. The degree of this circulatory disturbance is in proportion to the extent to which the sympathetic

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connections with the spinal cord are cut off. These fibers supplying the splanchnic area usually emerge from the cord from about the second thoracic to the first lumbar segments, hence it follows that the circulatory disturbance will be less the lower the puncture is made.

What happens to the *pulse rate* we should be able to prophesy. The accelerator stimuli in the thorax being removed, the vagus acts and the pulse rate remains the same or falls even below the usual normal. The heart is actually beating easier since it has no volume of peripheral blood against which it must exert its force. That is why patients with cardiac symptoms do particularly well during the interim of anesthesia.

The *respirations* are likewise slowed down for the same reason as is the pulse. They are usually easier and freer; former dyspnea of circulatory origin frequently is entirely replaced by unlabored shallow breathing.

The vagus, supplying motor fibers to the abdominal contents, has a chance to display motor stimuli there in the absence of the sympathetic control. The intestines contract and frequently the rectal sphincter gives way and allows an escape of intestinal gases and fecal material if the intestines have not previously been emptied. This is of decided advantage in abdominal operations, and is sometimes made use of therapeutically in the treatment of ileus. The stomach is also excited to greater motor activity, and hence we see frequent nausea and sometimes vomiting a few minutes after the anesthesia has been administered.

The vasomotor shock encountered is not met with in other forms of anesthesia. While most authors have noted this reaction there is an inclination to overlook it as a thing of little gravity or concern. It is obvious that an alteration of so vital a function as the vasomotor tone offers a potential if not a real hazard.

Respiration may be still further and more seriously affected in spinal anesthesia. The amount of disturbance is again dependent upon the height reached by the anesthetizing fluids. If the lower lumbar segments alone are anesthetized there may be nothing more than the cessation of the respiratory movements of the abdominal muscles. The patient breathes slowly and shallowly, but does not suffer, since the diaphragm and thoracic movements carry on the respiratory act without difficulty. But if the anesthesia is carried to higher levels, then the chest muscles become paralyzed and distinct respiratory difficulties are encountered. If the anesthesia is carried to the level of the fourth cervical, then the phrenics are paralyzed as well and asphyxia promptly follows. Only efficient artificial respiratory methods will keep the patient alive, and these must be continued until the anesthesia is passed. Danger of respiratory paralysis probably offers the most serious menace to this form of anesthesia; it is probably responsible for the largest percentage of casualties.

The *functions* of the *skin* are little modified by the anesthetic so long as the vasomotor paralysis is prevented. With a drop in blood pressure there are noted a blanching of the skin and a profuse outpouring of sweat.

Uterine contractions are not abolished, but are usually lessened in intensity but not in frequency. The uterus contracts after delivery as rapidly as with other forms of anesthesia.

Hemorrhage is less with spinal anesthesia than with other forms. This is sometimes held as an argument against its use. The usually lessened blood pressure produces a partial ischemia during the operation, and with the restoration of blood pressure the tissues again regain their normal circulation. It is argued that this will promote postoperative hemorrhage. In obstetrical work this fact can be utilized to advantage, since oxytocics will keep the uterus contracted after delivery, and the net loss of blood is actually less than with other forms of anesthesia.

The duration of the anesthesia is dependent upon the quantity of anesthetizing drug used. The maximum effect of the drug is reached within fifteen to twenty minutes after injection after which there is a gradual recession, the upper levels returning to normal sensation first. The average dose gives anesthesia lasting from one to one and one-half hours.

SELECTION OF DRUG AND PATIENT

Choice of Drug.—Many drugs have been used for this type of anesthesia. In recent times the number has narrowed down to three or four. Cocain, stovain, tropacocain and novocain are those most commonly used. J. Ralston Wells [2] uses an especially prepared anhydrous cocain. He states that it affects only the sensory nerves, and when introduced into the spinal fluid interrupts the conductivity of the posterior roots and leaves the anterior roots unaffected. He states further that the entire body, including the head, can be safely rendered insensible to pain. He, however, also acknowledges that 50 per cent of patients were nauseated or vomited and that abrupt declines in blood pressure were frequent. These facts would speak for sympathetic paralysis. The fact that it does not affect the motor nerves would argue against its use where muscular relaxation is desired. Stovain is a powerful anesthetic and deeply paralyzes the motor nerves; it is likewise admittedly the most toxic, most actively hemolytic and the strongest protoplasmic poison. Its use is restricted to those who have thoroughly familiarized themselves with its toxicity through long handling. Tropacocain is less active as an anesthetic. Novocain is the drug of choice in most clinics. It is nonhemolytic, is less toxic and can be used in large doses with reasonable safety. We have used "Neocain" (Rachi-Neocaine Corbiere), a highly refined novocain manufactured by Corbiere and Lionnet Laboratories, Paris. In our experience it has shown a low degree of toxicity, uniform results and convenience for hospital or office work.

Dosage.—We have used the above drug in ampoules of 0.10 and 0.12 grams (1½ and 2 grains), depending upon the size of the patient. No untoward results have been noted which could be

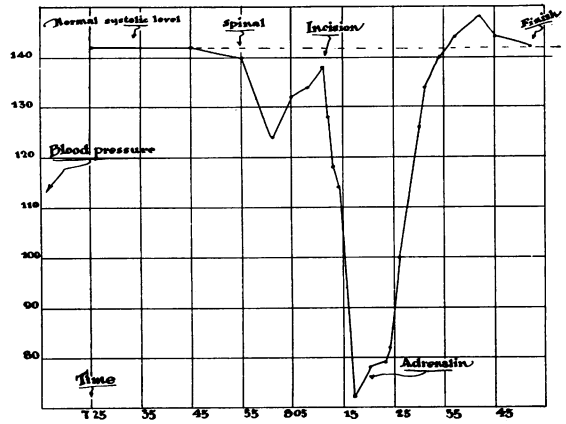

Fig. 1. Spinal Anesthesia without Ephedrin.—Inguinal hernia repair. Male, age fifty-eight, Portuguese. Typical curve showing vasomotor collapse after spinal anesthesia. Note very abrupt drop of systolic pressure to 72. Patient complained of weakness, vertigo, heat and nausea. Just before adrenalin was given, the radials were pulseless. Ephedrin would undoubtedly have prevented this distressing complication. (Note Fig. 2.)

attributed to the size of the dose. We have not used it in children for reasons noted below.

Choice of Patient.—Hypotension is universally considered a contraindication to spinal anesthesia; it is undoubtedly the major one. A systolic pressure of 110 is probably a safe minimum. Pulse pressure reading should also be considered; thus a patient with a systolic pressure of 130 but a diastolic reading of only 60, suggests lack of resistance. Those patients suffering from hypotension due to shock should be treated for shock before spinal anesthesia is attempted. *Obesity,* especially in those of stocky frame and short, thick neck, gives added peril to any form of anesthesia including spinal administration. *Children* make poor subjects for spinal anesthesia because of the difficulty in technique and their inability to coöperate after anesthesia has been established. *Deformity of the spinal column* may offer a mechanical difficulty. *Extremely nervous or hysterical* patients may prove a source of worry even after anesthesia has been established because of their inability or refusal to coöperate. Marked *cardiac interference* such as comes from pericarditis, advanced myocarditis, mediastinal tumors, cardiac displacements or pleuritic effusions, are poor subjects. Likewise those with noticeable *diaphragmatic interference* such as is found in extensive ascites, large intra-abdominal tumors or extensive pleuritic effusions, are also poor risks. *Convulsions* such as are found in tetanus, eclampsia or even hysterical convulsions, call for other forms of anesthesia, at least until spinal anesthesia can be established.

VARIOUS SITUATIONS TO AVOID

Insufficient Anesthesia.—This is not a serious circumstance. It may be due to insufficient dosage, but more often to a failure to introduce the drug into the subarachnoid space; occasionally some of the drug is lost through the point of needle puncture. A second injection is not wise; it is better to supplement the anesthesia obtained with inhalation methods.

Breaking the Needle.—This is a rare accident; it may be obviated by proper choice of patient who will coöperate. Platinum needles are more flexible than steel needles.

Circulatory Depression. — This we have observed in cases where measures were not used to prevent its happening. We do not agree with most authors that a vasomotor collapse during an operation is a harmless situation. Reasoning in terms of physiology and of surgical experience it is reasonable to conclude that any interference with so vital a function as vasomotor tone offers a potential menace to the patient's safety. It has been our observation that vasomotor collapse introduces a train of symptoms such as profuse perspiration, faintness, nausea and vomiting, air hunger and anxiety on the part of the patient, and usually also the operator. In those cases in which we have been able to sustain the normal systolic level we have not encountered these symptoms. Various authors have attempted to do this by repeated injections of adrenalin or the giving of a continuous intravenous saline-adrenalin infusion. Ockerbald and Dillon[3] of Kansas City first suggested the use of ephedrin for this purpose. They gave the drug when the blood pressure had dropped 10 per cent, and noted favorable results. We have used the drug in a small series of cases and find that its effect is much more lasting than adrenalin, that the drug itself produces no untoward symptoms, and when given at the time of spinal injection, will maintain the systolic level throughout a long operation and thus eliminate entirely this universal source of danger which accompanies spinal anesthesia. In those cases where the drug was not used at the time of spinal injection and an abrupt drop of blood pressure was noted, we have been able to demonstrate the supportive effect of the drug within a few minutes after injection. We feel that blood pressure readings at two to five-minute intervals will give the best index of the patient's condition and should be done routinely during spinal anesthesia. We have used doses of 0.1 grams of ephedrin sulphate put up in sterile ampoules. These prove most effective when given (subcutaneously) at the time of spinal injection.

Respiratory Depression.—In our small series of cases we have not encountered this complication except the mild air hunger noted when blood pressure had dropped to low ebb. It is the observation from most clinics that those who proved casualties during the operation under spinal anesthesia did so because of the gravity of the surgical condition, or because of respiratory failure. To guard against respiratory depression calls for close attention to several details of this mode of anesthesia.

TECHNIQUE IN PREPARATORY TREATMENT AND INJECTION

Preliminary preparation includes the administration of a narcotic before operation. Most operators still adhere to the use of scopolamin in

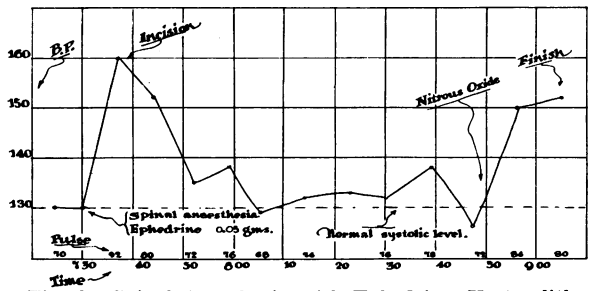

Fig. 2. Spinal Anesthesia with Ephedrin.—Ureterolithotomy. Japanese, female, age forty. Here 0.05 grams. Ephedrin was given at time of spinal anesthesia. Note how systolic pressure level was kept above normal throughout operation. Nitrous oxid inhalations supplemented spinal anesthesia during last ten minutes. Patient was perfectly comfortable throughout.

conjunction with morphin. We are under the impression that there are a fairly large number of patients who do not show the desired effect after a dose of scopolamin; they show motor excitation rather than a lowering of motor threshold impulses. We have used morphin-atropin combination with better satisfaction. The morphin depresses respiration, but the atropin is a respiratory stimulant and also lessens the secretions of the throat and helps thus to keep the air passages free. We have seen no advantage in giving it in divided doses; a single hypodermic dose one-half hour before operation has been our method. We also feel that there is some value in letting a patient face so important an event as a major operation with some food in his stomach. It does not seem rational to rob a patient of that stimulus which most American people get from their breakfast coffee or tea. For the purpose of bolstering up the patient's morale as well as for the unquestionable value of coffee as a respiratory and cardiac stimulant, we routinely give coffee or tea without milk one and one-half hours before operation, regardless of the type of anesthesia we have chosen. It does not interfere with the anesthetic, is definitely supportive, and certainly increases the feeling of well-being for the patient. *Point of injection* is not to be selected without recalling the dangers to be encountered when the thoracic and cervical segments become anesthetized. We have called attention to the fact that the spinal cord itself is little influenced by the anesthetizing solutions; hence the old supposition that respiratory paralysis was due to the drug reaching the vital centers in the medulla does not hold true. We feel that respiratory paralysis is occasioned by a succession of muscles of respiration being thrown out of function by the anesthetizing drug, and when finally the level of anesthesia is high enough to involve the phrenics coming from the fourth cervical region, respiratory paralysis becomes complete. It follows, then, that injections should be made low enough to avoid anesthetizing these higher levels. We have confined our operative work under this form of anesthesia to the abdomen, pelvis, rectum and lower extremities, and have encountered no respiratory difficulties of moment. In doing this work we have not gone higher than the twelfth thoracic region. The *amount of fluid withdrawn* has a definite influence on the level to which the anesthetizing fluid will rise above the point of injection. Reasoning in terms of physiology again, we have not conceded the *rationale* of withdrawing more fluid than is necessary to dissolve the drug. We usually withdraw 2 to 3 cc. of the fluid and again replace it after the drug has been dissolved. This tends to keep the cerebrospinal fluid pressure at its self-adapted level. We can see only danger in the practice of withdrawing excess fluids, thereby creating an area of negative pressure which allows anesthetizing solutions to soar to dangerously high levels after injection and thus court respiratory difficulties. The *pressure of injected fluids* also is worthy of attention. Some authors feel that they can control the level of anesthetic influence by the amount of pressure they apply to the fluids as they are introduced into the subarachnoid space. Since the cerebrospinal fluid pressure is not the same in each person, and since the variability of pressure exerted by non-mechanical means is subject to wide ranges, we feel that this practice carries with it some risks. We have followed the practice of injecting fluids very slowly in an attempt to establish a regional zone of block at the point of injection, and not to send currents and eddies of novocain solution through distant regions of the subarachnoid space. The *position of the patient* is of importance. Novocain is *very* quickly absorbed. We make it a rule to put the patient into a Trendelenburg position immediately after the spinal injection; they are kept with the head lowered for twenty-four hours after the operation. Labat [4] emphasizes the importance of this procedure. Immediately the question comes up whether the novocain does not flow toward the cervical region and thus paralyze higher levels of the cord. Labat has demonstrated that it does not; repeated withdrawals of fluid above the point of injection failed to reveal any trace of novocain. Contrary to all former notions we feel that the Trendelenburg position is the most logical one, and have encountered no difficulties in using it routinely. In general it may be stated, that to avoid respiratory complications one needs only to guard against applying the anesthetic solutions to those areas where they can interrupt the function of the muscles of respiration. It need hardly be stated that if there should develop a cessation of respiration, diligent and thorough artificial respiratory methods must be resorted to until such time as the anesthetic has worn off.

SUMMARY

The technique of spinal anesthetics has been developed to a high state of refinement and offers less risk than other forms of anesthesia for *surgical work below the diaphragm.* An understanding of the physiological background and a close attention to details of procedure should allow the operator of less extensive experience to utilize this form of anesthesia to a life-saving advantage. The maintenance of normal blood-pressure level by the use of ephedrin and avoidance of respira-

tory difficulties by keeping the anesthetizing drugs below the thoracic levels, should materially increase the safety of the patient.

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REFERENCES

1. Babcock, W. W.: Regional Anesthesia, Surg., Gynec. & Obst., xxxvii, 3, September, 1923.

2. Wells, J. Ralston: Spinal Anesthesia with Anhydrous Cocain, Ann. Surg., lxxxv, May, 1927.

3. Ockerbald, Nels F., and Dillon, T. G.: J. A. M. A., lxxxvii, 15, April 9, 1927.

4. Labat, Gaston: Circulatory Disturbances Associated with Subarachnoid Nerve-Block, Long Island Medical Journal, xxi, 10, October, 1927.

CARBON MONOXID POISONING*

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DISCUSSION *by Morton R. Gibbons, M. D., San Francisco; William C. Hassler, M. D., San Francisco; Alex. M. Lesem, M. D., San Diego.*

CARBON MONOXID is the most widespread and important toxic agent of modern civilization, preëminently greater than classic plumbism, on account of its multitude of applications in industry. The significance of this hazard warrants an intense interest of the medical profession and the general public on account of the incidence of morbidity and mortality from the use of illuminating or heating gas for domestic purposes and the deadliness of exhausts from automobiles and other gas engines.

SOURCES OF MODERN DANGER

The very general use of automobiles and other gasoline-burning engines whose exhausts contain 7 per cent of carbon monoxid, and the consequent vitiation of atmosphere in garages, traffic tunnels, and congested thoroughfares, has given public health and industrial hygienists much concern. A recent average of 141 tests made by United States Public Health chemists in city streets at peak hours of traffic showed a contamination of 0.8 in 10,000 parts of air, while in 102 tests made in twenty-seven garages taken in fourteen different cities the average carbon monoxid content was 2.1 parts in 10,000. One of the greatest dangers to life is encountered in warming up an engine in the small, closed garage; a hazardous act that should be given universal publicity. Coroner reports annually record the increased fatalities as a result of idling a running motor in a garage. It has been estimated that a motor will discharge two feet of carbon monoxid every minute, so that in twenty minutes the atmosphere would be concentrated enough to kill a dog by asphyxiation.

From an industrial hygienist's view, the amount of carbon monoxid should not be more than one part in 10,000 parts of air where workers are employed. Every industrial physician and employer should be familiar with the approximate amount of carbon monoxid eliminated in the special processes of manufacturing at his plant. Whenever carbon-containing fuels are burned, such as coal, gasoline, oil or charcoal, without sufficient oxygen completely to oxidize the fuel, carbon monoxid is liberated. The various industrial processes, whether accomplished by furnaces of all kinds, explosions, blasting, gas works, heating, and oil-distilling plants, furnish many cases of poisoning from this colorless, tasteless, and odorless gas.

DIAGNOSIS

The diagnosis of carbon monoxid poisoning rests mainly on a history of possible exposure and the presence of carbon monoxid in the blood either before or after death. At autopsy, when evidence is observed of the preservation of the body from decomposition—the cherry-red color of the organs, tissues, and blood, and the fluidity of the latter—there is no question of the diagnosis, CO poisoning. It is important to note that the greatest changes in the tissues found at postmortem are those of extensive fatty degeneration of the heart, kidney, walls of blood vessels, and other tissues. Minute hemorrhages throughout the brain are common. This one poisonous feature of carbon monoxid, *i. e.* the rapid fatty degeneration of organs, may have some bearing on the increase in and explanation of the mortality statistics of heart disease.

Every industrial precaution to detect carbon monoxid should be studied and observed. Normally the gas is odorless, except when mixed with other gases as in the case of common illuminating gas, which contains 30 per cent CO. In mines canary birds or mice are used to detect dangerous atmospheres. These animals are twenty times more susceptible than man. A bird will show signs of distress when exposed for one hour to 0.1 per cent of CO.

The Bureau of Mines has developed a detector which will give immediate positive results with carbon monoxid in the air in concentration of .07 per cent or more. This is known as hoolamite or the activated iodin pentiodid indicator, perfected by Teague, and a proven instrument of great value in mine rescue work and in testing plants where this hazard prevails.

Sayers and Yant of the United States Bureau of Mines have developed a quick and accurate method for the quantitative determination of carbon monoxid in the blood and air by the pyrotannic acid test. It is of great value in making a positive diagnosis in suspected cases. Normal diluted blood that has been shaken with an equal volume of one per cent tannic acid produces a gray suspension; whereas blood containing carbon monoxid remains carmin red. A color index is made by treating dilutions of varying strengths of blood with tannic acid. A sample of unknown blood prepared in similar strength could be readily matched with these corresponding standards, and the percentage of carbon monoxid ascertained.

The symptoms of carbon monoxid may be divided into two stages. In the first stage there is a feeling of tightness across the forehead, dizziness, frontal and basal headache, smarting of the eyes, lack of proper muscular coördination, nau-

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